

WEST Search History

DATE: Monday, December 18, 2006

Hide?	<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>
		<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=AND</i>	
<input type="checkbox"/>	L3	L2 and (streptococcus or streptococcal or (group adj1 b))	26
<input type="checkbox"/>	L2	glyceraldehyde same phosphate same dehydrogenase same NADP	113
<input type="checkbox"/>	L1	6800744.pn.	2

END OF SEARCH HISTORY

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES

PLEASE LOGON:

ENTER PASSWORD:

Welcome to DIALOG

Dialog level 05.15.00D

Last logoff: 09dec06 14:21:17

Logon file405 18dec06 16:48:54

*** ANNOUNCEMENTS ***

NEW FILES RELEASED

***Engineering Index Backfile (File 988)

***Verdict Market Research (File 769)

***EMCare (File 45)

***Trademarkscan - South Korea (File 655)

RESUMED UPDATING

***File 141, Reader's Guide Abstracts

RELOADS COMPLETED

***Files 340, 341 & 942, CLAIMS/U.S. Patents - 2006 reload now online

***Files 173 & 973, Adis Clinical Trials Insight

***File 11, PsycInfo

***File 531, American Business Directory

DATABASES REMOVED

***File 196, FINDEX

***File 468, Public Opinion Online (POLL)

Chemical Structure Searching now available in Prous Science Drug

Data Report (F452), Prous Science Drugs of the Future (F453),

IMS R&D Focus (F445/955), Pharmaprojects (F128/928), Beilstein

Facts (F390), Derwent Chemistry Resource (F355) and Index Chemicus

(File 302).

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* * *

SYSTEM:HOME

Cost is in DialUnits

Menu System II: D2 version 1.8.0 term=ASCII

*** DIALOG HOMEBASE(SM) Main Menu ***

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

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/H = Help

/L = Logoff

/NOMENU = Command Mode

Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database (e.g., B1 for ERIC).

? b 410

```
18dec06 16:48:54 User226352 Session D977.1
$0.00      0.240 DialUnits FileHomeBase
$0.00 Estimated cost FileHomeBase
$0.00 Estimated cost this search
$0.00 Estimated total session cost  0.240 DialUnits
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File 410:Dialog Comm.-of-Interest Newsl/Jul (c) 2006 Dialog

Set	Items	Description
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? set hi ;set hi

HIGHLIGHT set on as ''

HIGHLIGHT set on as ''

? b biochem

>>> 76 is unauthorized

>>>1 of the specified files is not available

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18dec06 16:49:09 User226352 Session D977.2
$0.00      0.115 DialUnits File410
$0.00 Estimated cost File410
$0.06 TELNET
$0.06 Estimated cost this search
$0.06 Estimated total session cost  0.356 DialUnits
```

SYSTEM:OS - DIALOG OneSearch

File 5:Biosis Previews(R) 1969-2006/Dec W2

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File 6:NTIS 1964-2006/Dec W1

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File 24:CSA Life Sciences Abstracts 1966-2006/Oct

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File 34:SciSearch(R) Cited Ref Sci 1990-2006/Dec W2

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File 40:Enviroline(R) 1975-2006/Oct

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File 50:CAB Abstracts 1972-2006/Nov

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File 65:Inside Conferences 1993-2006/Dec 15

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File 71:ELSEVIER BIOBASE 1994-2006/Dec W3

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File 73:EMBASE 1974-2006/Dec 18

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File 94:JICST-EPlus 1985-2006/Sep W1

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File 98:General Sci Abs 1984-2006/Dec

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File 103:Energy SciTec 1974-2006/Sep B1

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File 136:BioEngineering Abstracts 1966-2006/Oct

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 File 143: Biol. & Agric. Index 1983-2006/Nov
 (c) 2006 The HW Wilson Co
 File 144: Pascal 1973-2006/Nov W4
 (c) 2006 INIST/CNRS
 File 155: MEDLINE(R) 1950-2006/Dec 06
 (c) format only 2006 Dialog
 *File 155: MEDLINE has temporarily stopped updating with UD=20061206.
 Please see HELP NEWS154 for details.
 File 156: ToxFile 1965-2006/Nov W1
 (c) format only 2006 Dialog
 *File 156: ToxFile has stopped updating with MEDLINE records. Please
 see HELP NEWS 154 for details.
 File 162: Global Health 1983-2006/Nov
 (c) 2006 CAB International
 File 172: EMBASE Alert 2006/Dec 18
 (c) 2006 Elsevier B.V.
 File 305: Analytical Abstracts 1980-2006/Dec W3
 (c) 2006 Royal Soc Chemistry
 *File 305: Alert feature enhanced for multiple files, duplicate
 removal, customized scheduling. See HELP ALERT.
 File 369: New Scientist 1994-2006/Sep W4
 (c) 2006 Reed Business Information Ltd.
 File 370: Science 1996-1999/Jul W3
 (c) 1999 AAAS
 *File 370: This file is closed (no updates). Use File 47 for more current
 information.
 File 393: Beilstein Abstracts 2006/Q4
 (c) 2006 Beilstein GmbH
 File 399: CA SEARCH(R) 1967-2006/UD=14524
 (c) 2006 American Chemical Society
 *File 399: Use is subject to the terms of your user/customer agreement.
 IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.
 File 434: SciSearch(R) Cited Ref Sci 1974-1989/Dec
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Set	Items	Description
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? s	group(w)B	or agalactiae
	Processing	
	Processed	10 of 27 files ...
	Completed processing	all files
	7600127	GROUP
	6689946	B
	175350	GROUP(W)B
	24619	AGALACTIAE
S1	189467	GROUP(W)B OR AGALACTIAE
? s s1	and glycleraldehyde	and phosphate and dehydrogenase and NADP
	189467	S1
	0	GLYCLERALDEHYDE
	1501841	PHOSPHATE
	777269	DEHYDROGENASE
	73527	NADP
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	1501841	PHOSPHATE
	777269	DEHYDROGENASE
	73527	NADP
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	49467	GLYCERALDEHYDE
	1501841	PHOSPHATE
	777269	DEHYDROGENASE

73527 NADP
S4 2171 GLYCERALDEHYDE AND PHOSPHATE AND DEHYDROGENASE AND NADP
? s s1 and s4
189467 S1
2171 S4
S5 3 S1 AND S4
? rd s5

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.

S6 2 RD S5 (unique items)

? t s6/7/all

>>>Format 7 is not valid in file 143

6/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS Previews(R)
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0009577908 BIOSIS NO.: 199598045741
Effect of vitamin D deficiency and 1,25-dihydroxyvitamin D-3 on rat heart
metabolism
AUTHOR: Stio Maria; Lunghi Barbara; Maria Teresa Iantomasai; Vincenzini
Teresa; Treves Cristina (Reprint)
AUTHOR ADDRESS: Dep. Biochem. Sci., Univ. Florence, Viale Morgagni 50,
50134 Florence, Italy**Italy
JOURNAL: Journal of Molecular and Cellular Cardiology 26 (11): p1421-1428
1994 1994
ISSN: 0022-2828
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The purpose of this study was to investigate whether vitamin D-3 deficiency and 1,25-dihydroxyvitamin D-3 treatment affect some aspects of heart metabolism in the rat. To this end, five experimental groups were studied: (1) the control group of the vitamin D-3 supplemented rats (Group A); (2) rachitic rats (Group B); (3) rachitic rats treated with 1,25-dihydroxyvitamin D-3 (Group C); (4) rats fed a vitamin D-deficient diet (Group D); (5) rats fed a vitamin D-deficient diet and treated with 1,25-dihydroxyvitamin D-3 (Group E). The five groups were compared by checking in the heart some metabolic parameters, i.e. citrate content, and enzyme activities in cytosol and mitochondria. Citrate content was higher in the heart of treated animals when compared with the control. As regards the enzymatic activities in heart mitochondria, NAD+-dependent isocitrate dehydrogenase remarkably decreased in Group B rats and 1,25-dihydroxyvitamin D-3 restored quite normal values. NADP+-dependent isocitrate dehydrogenase decreased in Group B and Group D animals, and 1,25-dihydroxyvitamin D-3 treatment was effective in restoring control values. Cytochrome c oxidase activity did not change, while citrate synthase showed an increase in all the treated rats. As regards the cytosolic enzymes, fructose-6-phosphate kinase increased in the two groups of vitamin D-deplete rats in comparison with the control. Glyceraldehyde-3-phosphate dehydrogenase and 3-phosphoglycerate kinase showed a similar trend: an increase in all the treated animals. In heart homogenate, acylphosphatase and acid phosphatase activities were also determined. Acylphosphatase increased in the treated rats, while acid phosphatase decreased in the rats injected with 1,25-dihydroxyvitamin D-3. These results support the hypothesis of a participation of 1,25-dihydroxyvitamin D-3 in some aspects of heart metabolism.

6/7/2 (Item 1 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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03613787 Genuine Article#: PQ983 Number of References: 35

Title: EFFECT OF VITAMIN-D DEFICIENCY AND 1,25-DIHYDROXYVITAMIN-D-3 ON
RAT-HEART METABOLISM

Author(s): STIO M; LUNGHI B; IANTOMASI T; VINCENZINI MT; TREVES C

Corporate Source: UNIV FLORENCE, DEPT BIOCHEM SCI, VIALE MORGAGNI 50/I-50134
FLORENCE//ITALY//; UNIV FLORENCE, DEPT BIOCHEM SCI/I-50134
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Journal: JOURNAL OF MOLECULAR AND CELLULAR CARDIOLOGY, 1994, V26, N11 (NOV)
, P1421-1428

ISSN: 0022-2828

Language: ENGLISH Document Type: ARTICLE

Abstract: The purpose of this study was to investigate whether vitamin D-3 deficiency and 1,25-dihydroxyvitamin D-3 treatment affect some aspects of heart metabolism in the rat. To this end, five experimental groups were studied: (1) the control group of the vitamin D-3 supplemented rats (Group A); (2) rachitic rats (Group B); (3) rachitic rats treated with 1,25-dihydroxyvitamin D-3 (Group C); (4) rats fed a vitamin D-deficient diet (Group D); (5) rats fed a vitamin D-deficient diet and treated with 1,25-dihydroxyvitamin D-3 (Group E). The five groups were compared by checking in the heart some metabolic parameters, i.e. citrate content, and enzyme activities in cytosol and mitochondria. Citrate content was higher in the heart of treated animals when compared with the control. As regards the enzymatic activities in heart mitochondria, NAD(+)-dependent isocitrate dehydrogenase remarkably decreased in Group B rats and 1,25-dihydroxyvitamin D-3 restored quite normal values. NADP (+)-dependent isocitrate dehydrogenase decreased in Group B and Group D animals, and 1,25-dihydroxyvitamin D-3 treatment was effective in restoring control values. Cytochrome c oxidase activity did not change, while citrate synthase showed an increase in all the treated rats. As regards the cytosolic enzymes, fructose-6-phosphate kinase increased in the two groups of Vitamin D-deplete rats in comparison with the control. Glyceraldehyde-3-phosphate dehydrogenase and 3-phosphoglycerate kinase showed a similar trend: an increase in all the treated animals. In heart homogenate, acylphosphatase and acid phosphatase activities were also determined. Acylphosphatase increased in the treated rats, while acid phosphatase decreased in the rats injected with 1,25-dihydroxyvitamin D-3. These results support the hypothesis of a participation of 1,25-dihydroxyvitamin D-3 in some aspects of heart metabolism.

? ds

Set	Items	Description
S1	189467	GROUP(W)B OR AGALACTIAE
S2	0	S1 AND GLYCLERALDEHYDE AND PHOSPHATE AND DEHYDROGENASE AND NADP
S3	0	GLYCLERALDEHYDE AND PHOSPHATE AND DEHYDROGENASE AND NADP
S4	2171	GLYCERALDEHYDE AND PHOSPHATE AND DEHYDROGENASE AND NADP
S5	3	S1 AND S4
S6	2	RD S5 (unique items)
?		

---Logging off of Dialog---

? logoff

18dec06 16:53:34 User226352 Session D977.3
\$2.74 0.457 DialUnits File5
\$2.20 1 Type(s) in Format 7
\$2.20 1 Types
\$4.94 Estimated cost File5

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	\$6.82	1 Types		
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\$1.33	TELNET			
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\$38.96	Estimated total session cost 3.761 DialUnits			

Logoff: level 05.15.00 D 16:53:34



BSCI 424 — PATHOGENIC MICROBIOLOGY —
Fall 2000



Streptococcus Summary

This Page is Under Construction



[Click Here to View More Images of Streptococci](#)

Morphology & Physiology:

● General Characteristics:

- Gram-Positive, Nonmotile Cocci (0.5-1.2um) often Arranged in Pairs or Chains (see [WebLinked image](#); see [WebLinked image](#))
- Exist as Commensals and Parasites for Man, Animals or Saprophytes
- Most are facultative anaerobes
- Complex nutritional requirements (fastidious): Require blood- or serum-enriched media; Inability to synthesize many basic building blocks
- Fermentative metabolism: Carbohydrates are fermented without the production of gas; Major endproducts are lactic acid, ethanol, acetate
- Catalase negative: Useful to distinguish streptococci from staphylococci
- Oxidase negative
- Rigid cell wall with typical Gram-positive peptidoglycan layer, inner plasma membrane, mesosomal vesicles, nucleoid; Divide by crosswall septation
- Some strains have hyaluronic acid capsule; Produce hyaluronidase later in

growth cycle

● Cellular Structure of Group A Streptococci: *S. pyogenes*

- Cell wall contains group- and type-specific antigens
 - Group-specific carbohydrate (C-polysaccharide):
 1. ~10% dry weight of the cell
 2. Branched polymer of L-rhamnose and N-acetyl-D-glucosamine (2:1 ratio); latter is antigenic component; linked by phosphate-containing bridges to peptidoglycan which is composed of N-acetyl-D-glucosamine, N-acetyl-D-muramic acid, D-glutamic acid, L-lysine, and D- and L-alanine
 - Type-specific proteins: Two major classes, M and T antigens; Minor classes include: F, R and M-like or M-associated proteins (MAP) antigens
 1. M Protein: Fimbriae-like, "hairy" extensions associated with virulent strains
 - Resistant to heat and acid; Trypsin sensitive
 - Carboxy (-COOH) terminal end of molecule is anchored in cell wall and sequence is highly conserved among all M serotypes
 - More than 80 M serotypes are due to antigenic diversity of the M protein's amino (-NH₂) terminus which is surface-exposed
 2. T protein: Useful epidemiological marker; Not identified as virulence factor
 - Trypsin resistant; Resistant to heat and acid
 3. M-like proteins: Structurally similar to M protein
 4. F protein: Binds fibrinogen
 5. Lipoteichoic acid is also antigenic
- Capsular polysaccharide: composed of hyaluronic acid (also found in host connective tissue)

Classification Schemes (Taxonomy):

● Based on biochemical (Physiological) properties

● Based on clinical presentation:

- Pyogenic (suppurative)
- Oral
- Enteric

☹ Based on hemolytic patterns:

- ☹ Beta-hemolytic: beta-hemolysis often enhanced under anaerobic conditions
- ☹ Alpha-hemolytic
- ☹ Gamma-hemolytic

☹ Based on serology: Lancefield serogrouping of **beta-hemolytic streptococci**; Lancefield group-specific antigens

☹ Lancefield (1933) developed useful serotyping system for classification of beta-hemolytic streptococci based on the group-specific antigenic composition of cell wall carbohydrates (Note: viridans streptococci and *Streptococcus pneumoniae* have no group-specific antigen)

☹ Serogroups A through H and K through V

● Major pathogens

1. Group A Streptococci: *Streptococcus pyogenes*
2. Group B Streptococci: *S. agalactiae* (Note: May also be alpha-hemolytic or nonhemolytic)
3. *Enterococcus faecalis* and *E. faecium* were previously classified as Group D streptococci

● Common pathogens

1. Group C Streptococci: *S. equismilis* (pharyngitis); *S. anginosus* (abscess formation)
2. Group F Streptococci: *S. anginosus* (abscess formation)
3. Group G Streptococci: *S. anginosus* (abscess formation)

● Uncommon pathogens

1. Group D Streptococci: *S. bovis*, *S. durans*, *S. avium* (foodborne disease)
2. Groups E, H, and K through

☹ Streptococci lacking group-specific antigens

● Viridans streptococci: Gamma or alpha-hemolysis

1. *Streptococcus mutans* group
2. *S. sanguis* group
3. *S. salivarius* group
4. *S. mitis* group
5. *S. anginosus-milleri* group
6. *Streptococcus* spp.

● *Streptococcus pneumoniae* *

Clinical Syndromes:

● **Group A *Streptococcus* (*S. pyogenes*):** One of the most important human pathogens; Commonly associated with a diverse group of human diseases, including both suppurative (pus-forming) and nonsuppurative diseases

- **Suppurative streptococcal diseases (Acute streptococcal infection)**
 - **Pharyngitis (& tonsillitis):** "strep throat" --- Suffix "-itis" refers to inflammation of (in this case, inflammation of the pharynx or tonsils)
 - **Scarlet fever:** complication of streptococcal pharyngitis when infecting strain is lysogenized ; Frequently develop scarletina rash on upper chest spreading to extremities
 - **Cutaneous & soft tissue infections:**
 1. **Pyoderma** (Impetigo: contagious pyoderma with superficial yellow weeping lesions)
 2. **Erysipelas:** Acute superficial cellulitis of skin with lymphatic involvement; face and lower extremities, skin and subcutaneous tissues
 3. **Cellulitis:** Involvement of deeper subcutaneous tissues; Deeper invasion with systemic symptoms
 4. **Necrotizing fasciitis** (a.k.a., "flesh-eating bacteria"): Infection deep in subcutaneous tissues that spreads along fascial planes, destroying muscle and fat; Initially cellulitis followed by bullae (fluid filled blisters; bulla is singular), gangrene, systemic toxicity, multiorgan failure and mortality in more than 50% of patients
 5. **Streptococcal toxic shock syndrome:** Multisystem toxicity following soft tissue infection progressing to shock and organ failure (not to be confused with Staphylococcal Toxic Shock Syndrome where hyperabsorbent tampons have been identified as an important risk factor); Systemic infection following soft tissue infection
 - **Other suppurative diseases:** Puerperal sepsis (associated with childbirth); Lymphangitis (inflammation of lymphatic vessel(s)); Pneumonia
- **Bacteremia:** bacteria in the blood with mortality approaching 40%; **Septicemia** (sepsis): systemic disease associated with persistent presence of bacterial cells, bacterial toxins or other bacterial products in the blood
- **Acute nonsuppurative (non-pus-forming) sequelae (complications) of Group A streptococcal disease:** Post-infection sequelae
 - **Acute rheumatic fever (ARF):** Inflammation of heart, joints, blood vessels, subcutaneous tissues
 1. Nonsuppurative inflammatory reaction characterized by arthritis, carditis, chorea (disorder of nervous system with

involuntary spastic movements), erythema marginatum (skin redness with defined margin), or subcutaneous nodules

2. Morbidity and mortality linked to subsequent valvular heart disease
3. Poorly understood pathogenesis with several proposed theories including cross-reactivity of heart tissues and streptococcal antigens, exotoxins, or direct invasion

- **Rheumatic heart disease:** Chronic, progressive heart valve damage

- **Acute glomerulonephritis:** Acute inflammation of renal (kidney) glomeruli

1. Signs include dark, smoky urine with RBC's, RBC casts, white blood cells, depressed serum complement, decreased glomerular filtration rate
2. Granular accumulations of immunoglobulin due to deposition of immune complexes within the kidney

● Group B *Streptococcus* (*S. agalactiae*)

- **Neonatal disease** (Early onset and late-onset): Infection can occur in utero, at birth, or during first few months of life

- Neonatal sepsis (septicemia)
- Meningitis: Inflammation of the meninges
- Pneumonia

- **Obstetric complications:**

- Urinary tract infections (UTIs)
- Puerperal (postpartum) sepsis
- Amnionitis: Inflammation of amnion (membrane enveloping fetus)
- Endometritis: Inflammation of the endometrium (inner mucous membrane of the uterus)
- Wound infections

- **Infections in non-pregnant adults:** Primarily skin and soft tissue involvement

- Genitourinary tract infections (urosepsis)
- Wound infections
- Pneumonia
- Bacteremia

● Other Beta-Hemolytic Streptococci:

- Most commonly Group C (*S. equisimilis*, *S. anginosus*), Group F (also *S. anginosus*), and Group G (also *S. anginosus*)

- Pharyngitis
- Abscess formation
- Bacteremia
- ☹ Group D: Foodborne disease similar to staphylococcal intoxication

☹ Non-Beta-Hemolytic Streptococci: Viridans Streptococci (heterogeneous collection of alpha- and non-hemolytic).

- ☹ Dental caries (cavities): *S. mutans* adhere to enamel via production of insoluble dextran from glucose; Allows other organisms to adhere (plaque formation) with acid production causing tooth decay; *Lactobacillus* spp. cause caries of dentin secondary to enamel caries caused by mutans streptococci (see also *Actinomyces*)
- ☹ Subacute bacterial endocarditis: *S. mutans*, *S. sanguis* adhere to previously damaged heart valves via production of insoluble dextran from glucose
- ☹ Abscess formation (suppurative intraabdominal infections)

Epidemiology:

☹ General Overview:

- ☹ Streptococci have a predilection for the upper respiratory tract or skin; Strains that colonize the skin are usually different "antigenic types" from those that colonize the throat
- ☹ Group A streptococci commonly colonize the oropharynx of healthy children and young adults or the skin
 - Colonization is transient due to development of M protein-specific immune response
 - Rapidly killed after phagocytic ingestion, but cell walls that are not digested may lead to chronic inflammatory lesions
 - Elevated incidence of carriage
- ☹ Non-beta-hemolytic streptococci are competitive; Produce bacteriocins which are inhibitory to Group A strains

☹ Acute Streptococcal Infection: Disease from recently acquired strain

- ☹ Pharyngitis (peak ages 5-15 years) and scarlet fever (complication of streptococcal pharyngitis):
 - Transmitted person-to-person by droplets from respiratory secretions; Crowding increases risk (e.g., classrooms, day care facilities)
 - Acute rheumatic fever and glomerulonephritis sequelae to pharyngitis

- **Scarlet fever** results from infection with a strain that is lysogenized with a temperate bacteriophage that genetically encodes for pyrogenic exotoxin (formerly known as erythrogenic toxin or erythrotoxin)
- **Cutaneous & soft tissue infections:**
 - Transmitted through breaks in skin after direct contact with infected person, fomite, or arthropod vector
 - T-antigen typing useful where non-M typeable
 - Only glomerulonephritis sequelae
 - **Streptococcal toxic shock syndrome:** caused by different M serotypes than those causing pharyngitis; pyrogenic exotoxins, particularly exotoxin A, are produced
- **Neonatal and puerperal disease:**
 - Transient colonization of vagina observed in both pregnant and nonpregnant females, most commonly by limited number of serotypes
 - Colonize lower gastrointestinal (GI) tract and genitourinary tract
 - 60% of colonized mothers birth colonized babies; Neonatal disease in 3/1000 live births
 - Risk factors:
 1. Immune status of mother is more important risk factor than exposure to organisms for acquiring neonatal disease (maternal type-specific capsular antibodies transplacentally passed to fetus is protective during first few months of life as infant's immune system matures)
 2. Heavy vaginal colonization during birth increases likelihood of neonatal colonization, but does not increase risk of disease
 3. Complement deficiency
 4. Pregnancy risks include: Premature birth; Prolonged rupture of membranes; Fever during delivery
 - **Early onset neonatal disease:** Within 7 days of birth
 1. Acquired in utero or during delivery
 2. Three times more frequent than late onset disease
 3. 15-30% of post-meningeal infants experience **neurological sequelae** that include blindness, deafness, and severe mental retardation
 - **Late-onset neonatal disease:** Occurring 1 week to 3 months after birth
 1. Acquired from exogenous source: Mother, other infants, health care provider
 2. Presents most commonly as bacteremia with meningitis; Neurological complications are common

- **Puerperal sepsis:** Common cause of maternal death in pre-antibiotic era
- **Disease in nonpregnant adults**
 - Risk Factors:
 1. Diabetes mellitus
 2. Cancer
 3. Alcoholism
 - Proportionally more adult disease, but incidence is higher in neonates
- **Foodborne disease:** Infrequent foodborne disease outbreaks
 - Organisms multiply when contaminated foodstuffs are improperly refrigerated and allowed to stand at room temperature for several hours between preparation and consumption
 - Contamination of food is result of incomplete processing or unsanitary food handling by carriers (poor hygiene, ill or asymptomatic food handler) or unpasteurized milk (frequent outbreaks before advent of pasteurization)
 - High infectious dose of $>10^7$ organisms
 - Explosive common-source Group D outbreaks (2-36h incubation period) with a clinical syndrome similar to staphylococcal intoxication
 - Note: Septic sore throat, scarlet fever and other pyogenic and septicemic syndromes are also infrequently acquired from ingestion (low infectious dose of <1000 organisms) of contaminated food (onset after 1-3 days)
- **Sequelae of Acute Streptococcal Infection:** Serious suppurative and nonsuppurative sequelae in pre-antibiotic era; Nonsuppurative rheumatic fever and glomerulonephritis is still important in developing countries
- **Acute rheumatic fever (ARF)**
 - Within 2-3 weeks (latent period) following respiratory infection only (e.g., pharyngitis)
 - For diagnosis, must demonstrate recent infection by culture or serology; Detect an increase in antibody titer to at least one of SLO, DNase B, hyaluronidase, streptokinase
 - Following epidemic pharyngitis: ARF in as many as 3%; Following sporadic pharyngitis: ARF in 1 per 1000
- **Acute post-streptococcal glomerulonephritis**
 - Acute glomerulonephritis follows either respiratory (e.g., pharyngitis) or cutaneous streptococcal infection

- **Latent period:** 1-2 weeks after skin infection and 2-3 weeks after pharyngitis
 - Associated with specific, well-defined group of serotypes
 - Incidence following infection varies from less than 1% to 10-15%
 - Most often seen in children
- **Viridans Streptococcal Infections:** *Streptococcus mutans* is major dental pathogen
- **Acidoduric** (durable in acidic environment) and **acidogenic** (acid generating) properties, and the ability to synthesize extracellular adherent polysaccharides enable these organisms to establish initial colonization of dental surfaces and to provide a foundation layer for the formation of a complex **biofilm** known as dental plaque
 - Enamel caries is acid demineralization of enamel caused by lactic acid production by these bacteria during **fermentation** of sucrose or other carbohydrates and is dependent upon a variety of physical, chemical, and mechanical factors
 - Dental caries can be visualized as a disease state that occurs only when three factors or conditions co-exist. These three interdependent components are:
 - Status of host and teeth (sites that organisms can colonize, in particular solid surfaces and interfaces)
 - Specific microflora
 - Nutritional substrate (carbon and energy source)
 - **Gingivitis** and more severe periodontal diseases are a result of plaque-derived infection at the tooth and gum interface with loss of gum tissue and underlying bone
 - **Pyogenic oral infections** can be **acute** or **chronic** in nature, caused by mixed anaerobic infection of tissue by plaque flora, e.g., by trauma, invasive dental treatments, periodontal disease, or infected dental pulps

Pathogenesis & Immunity:

- **Lysogeny** (Lysogenic Conversion)
- Lysogenized **bacteriophages** may play key role in directing synthesis of various Group A streptococcal enzymes and toxins
- **Pyrogenic exotoxin** (a.k.a., erythrogenic toxin): Phage-associated muralysins produced by both Group A and C streptococci

☛ Grp C muralysin is N-acetylmuramyl-L-alanine amidase that lyses streptococcal cell walls; Used to produce L-forms (cells lacking rigid cell walls) or to purify membranes and M protein

☛ Virulence Factors of Group A *Streptococcus pyogenes*: Structural molecules; Toxins; Enzymes

☛ Cellular components

☛ Lipoteichoic acid

☛ Adherence to buccal epithelial cells

☛ Cytotoxic for wide variety of cells

☛ Colonization ligand: Forms complex network with M protein and binds via lipid moiety to fibronectin on epithelial cells

☛ M protein

☛ Antiphagocytic; alpha-helical coiled dimer; fibrillar molecule

☛ Inhibits complement component C3b (Factor H, serum control protein) deposition on streptococcal cell surface, inhibiting alternate complement pathway and opsonization

☛ Cell wall is potent activator of alternate complement pathway, but M-protein prevents these reactions

☛ Grp A Strep with type-specific M-protein survive until type-specific antibody response develops

☛ Amino-terminus (distal to cell) contains antigenically variable determinants; Three large tandem repeat regions contribute to antigenic diversity with intragenic recombination events

☛ Abnormal host immune response against cross-reactive antigens may result in sequelae

- ☉ **F protein:** Binds fibronectin of host; Major adhesin to throat and skin by mediating adherence to epithelial cells
- ☉ **Associated proteins:** Some under control of virulence regulon and co-regulated with M-protein
- ☉ C5a peptidase destroys chemotactic signals
- ☉ Serum opacity factor
- ☉ Fc receptor protein binds to Fc region of mammalian IgG
- ☉ Antiphagocytic capsular polysaccharide
- ☉ **Hyaluronic acid** mimics animal tissue ground substance found in host connective tissue and is therefore nonimmunogenic; assists in avoiding phagocytosis
- ☉ **Extracellular components**
 - ☉ **Hemolysins:** Two hemolytic and cytolytic toxins
 - ☉ Streptolysin O (SLO)
 - ☉ Prototype of oxygen labile or thiol-activated bacterial cytolytic protein toxins produced by *Streptococcus*, *Bacillus*, *Clostridium*, and *Listeria*
 - ☉ Irreversibly inactivated by cholesterol
 - ☉ Bind to cholesterol-containing membranes and form arc- or ring- shaped oligomers that make cell leaky (RBC's, PMN's, Platelets, etc.)
 - ☉ SLO induces rapid antibody response
 - ☉ SLO titer indicates recent infection (300-500 in pediatric populations)
 - ☉ Streptolysin S (SLS)

- ☉ Oxygen stable, non-antigenic
- ☉ Extractable only in presence of carrier or inducer, e.g., serum, albumin, RNase-resistant fraction of RNA
- ☉ Lytic for red and white blood cells and wall-less forms (protoplast, L- forms)
- ☉ Responsible for surface hemolysis on blood agar plates; Inhibited by phospholipids
- ☉ Pyrogenic exotoxins (a.k.a., erythrogenic toxins)
 - ☉ Produced by more than 90% of Grp A streptococci; 3 types Groups A, B, and C
 - ☉ Heat labile; Stable to acid, alkali, pepsin
 - ☉ Structural gene is carried by temperate bacteriophage, as is the case with diphtheria toxin
 - ☉ Result in fever and scarlet fever rash, increase susceptibility to toxic shock, cause reticuloendothelial system blockade, act as mitogens, myocardial and hepatic necrosis, decrease in antibody synthesis
 - ☉ Immunomodulators (superantigens) at low concentrations, stimulate T cells
 - ☉ Type C toxin increases permeability of blood-brain barrier to endotoxin and bacteria
- ☉ Nucleases: Reduce viscosity
 - ☉ Four antigenic types (A,B,C,D) in *S. pyogenes* assist in liquefaction of pus to generate growth substrates
 - ☉ Nucleases A, C have DNase activity

- Nucleases B, D also have RNase activity
- Require calcium and magnesium
- Antibody titers to DNase B useful serodiagnosis
- Other Enzymes
 - Two different streptokinases that catalyze conversion of plasminogen to plasmin, leading to digestion of fibrin (dissolve blood clots); may enable rapid dissemination through infected tissues
 - Hyaluronidase hydrolyzes hyaluronic acid; "spreading factor"
 - DPNase, cardiohepatic toxin, proteinase, NADase, ATPase, phosphatase, etc.
- Virulence Factors of Group B *Streptococcus agalactiae*
 - Enzymes of unknown pathogenicity: Dnases, hyaluronidase, neuraminidase, proteases, hippurase, hemolysins
- Immune Response Against Streptococcal Disease
 - Antibodies against type-specific capsular antigens are protective; Infants without type-specific maternal antibodies are at increased risk of acquiring infection
 - Bactericidal activity requires the presence of complement

Laboratory Identification

- General Overview
 - Gram stain of tissue sample can provide rapid initial diagnosis
 - Bacitracin sensitivity test for presumptively distinguishing between beta-hemolytic streptococci isolated from pharyngeal swabs (95% accuracy of Grp

A strep sensitivity)

⊕ Negative catalase used to differentiate from Staphylococcus

⊕ Group A Streptococci

⊕ Specimen Collection and Processing

⊕ Throat swabs from the posterior oropharynx (back of throat, e.g., tonsils) or from skin lesion

⊕ Microscopy: Gram stain

⊕ Cocci in short chains in clinical specimens; Longer chains seen in culture

⊕ Culture

⊕ Primary culture by pour and streak plate

⊕ Fastidious growth requirements; Optimal growth on enriched blood agar; Growth inhibited by glucose in medium (fermented with lactic acid buildup)

⊕ 1 to 2-mm domed white (grayish to opalescent) colonies with large zone of beta-hemolysis (several times that of diameter of colony) after 18-24h incubation

⊕ Colonies of encapsulated strains appear mucoid on moist media, wrinkled on dry media; Colonies of nonencapsulated strains appear small and glossy

⊕ Identification

⊕ Preliminary identification

⊕ Positive CAMP test

⊕ Hydrolysis of hippurate

⊕ Antigen detection

⊕ Group Specificity

⊕ Positive identification of **group-specific carbohydrate** (rhamnose, N-acetylglucosamine, galactose)

- Type Specificity
 - Type-specific capsular polysaccharide (sialic acid residue)
 - Type-specific proteins
- Methods
 - Bacitracin sensitivity
 - Rapid identification tests: Based on extraction of Grp A carbohydrate directly from throat swabs
 - Coagglutination
 - Fluorescent Antibody
 - Antigen detection
 - Detection of group-specific carbohydrate (dimer of N-acetylglucosamine & rhamnose) (formerly C-Polysaccharide)
 - Capillary tube precipitin tests
 - Variety of agglutination tests
 - Detection of M protein by Capillary Tube Precipitin Test with extracts or whole cells
 - Detection of T protein by Slide Agglutination Test with trypsin treated whole cells; Adjunct to M-typing; routine surveillance
 - Antibody Detection
 - ASO Test: Detection of antibodies against streptolysin O confirm a recent Group A streptococcal infection
 - Detection of DNase B
 - Group B Streptococci
 - Microscopy: Gram stain

- Cocci in short chains in clinical specimens; Longer chains in Cx
- Culture: Large, buttery-appearing colonies usually surrounded by a narrow zone of hemolysis

Treatment, Prevention & Control

- Therapy: Aimed at prevention of suppurative complications and the nonsuppurative sequelae of rheumatic fever and glomerulonephritis
- Group A Drug of Choice: Oral (PO) penicillin V (VK) or intramuscular (IM) benzathine penicillin; Alternatives: Erythromycin, clindamycin, cephalexin (oral cephalosporin)
- Group B Drug of Choice: Intravenous (IV) penicillin G; Alternate: Vancomycin
- High MIC (minimal inhibitory concentration)
- Antibiotic tolerance has been observed
- Tolerance: able to endure without effect
- Acute Rheumatic Fever
- Salicylates and corticosteroids for acute symptom reduction and control of long-term sequelae; Prevention by preventing initial strep infection or prompt treatment of pharyngitis
- Acute Post-Streptococcal Glomerulonephritis
- Therapy directed at secondary phenomenon of volume excess, hypertension, and seizures; Sodium restriction, diuretics, anticonvulsants
- Must demonstrate recent infection by culture or serology; Detect an increase in antibody titer to SLO or DNase B
- Renal biopsy, immunofluorescent exam, or electron microscopy to diagnose



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Search in ENZYME for: glyceraldehyde -3-phosphate dehydrogenase

Release of 12-Dec-2006

Please choose one of the following entries:

- 1.2.1.9 Glyceraldehyde-3-phosphate dehydrogenase (NADP(+)).
 (AN: Triosephosphate dehydrogenase.)
- 1.2.1.12 Glyceraldehyde-3-phosphate dehydrogenase (phosphorylating).
 (AN: GAPDH.
 NAD-dependent glyceraldehyde-3-phosphate dehydrogenase.
 Triosephosphate dehydrogenase.)
- 1.2.1.13 Glyceraldehyde-3-phosphate dehydrogenase (NADP(+)) (phosphorylating).
 (AN: NADP-dependent glyceraldehyde-3-phosphate dehydrogenase.
 Triosephosphate dehydrogenase (NADP(+)).
 Triosephosphate dehydrogenase (NADP+).)
- 1.2.1.59 Glyceraldehyde-3-phosphate dehydrogenase (NAD(P)(+)) (phosphorylating).
 (AN: NAD(P)-dependent glyceraldehyde-3-phosphate dehydrogenase.
 Triosephosphate dehydrogenase (NAD(P)(+)).
 Triosephosphate dehydrogenase (NAD(P)).)
- 1.2.7.6 Glyceraldehyde-3-phosphate dehydrogenase (ferredoxin).
 (AN: GAPOR.
 Glyceraldehyde-3-phosphate Fd oxidoreductase.
 Glyceraldehyde-3-phosphate ferredoxin reductase.)

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NiceZyme View of ENZYME: EC 1.2.1.9

Official Name	
Glyceraldehyde-3-phosphate dehydrogenase (NADP(+)).	
Alternative Name(s)	
Triosephosphate dehydrogenase.	
Reaction catalysed	
D-glyceraldehyde 3-phosphate + NADP(+) + H(2)O <=> 3-phospho-D-glycerate + NADPH	
Cross-references	
Biochemical	
Pathways; map number(s)	D5
PROSITE	PDOC00068
BRENDA	1.2.1.9
PUMA2	1.2.1.9
PRIAM enzyme-specific profiles	1.2.1.9
KEGG Ligand Database for Enzyme Nomenclature	1.2.1.9
IUBMB Enzyme Nomenclature	1.2.1.9
IntEnz	1.2.1.9
MEDLINE	Find literature relating to 1.2.1.9
MetaCyc	1.2.1.9
UniProtKB/Swiss-Prot	Q9SNX8.GAPN APIGR; Q1WIQ6.GAPN ARATH; Q43272.GAP P93338.GAPN NICPL; P81406.GAPN PEA; Q59931.GAP

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Search for

NiceZyme View of ENZYME: EC 1.2.7.6

Official Name	
Glyceraldehyde-3-phosphate dehydrogenase (ferredoxin).	
Alternative Name(s)	
GAPOR.	
Glyceraldehyde-3-phosphate Fd oxidoreductase.	
Glyceraldehyde-3-phosphate ferredoxin reductase.	
Reaction catalysed	
D-glyceraldehyde-3-phosphate + H ₂ O + 2 oxidized ferredoxin \rightleftharpoons 3-phospho-D-glycerate + 2 H ⁽⁺⁾ + 2 reduced ferredoxin	
Cofactor(s)	
Iron-sulfur; Tungsten-molybdopterin.	
Comment(s)	
<ul style="list-style-type: none"> • Thought to function in place of glyceraldehyde-3-phosphate dehydrogenase and possibly phosphoglycerate kinase in the novel Embden-Meyerhof-type glycolytic pathway found in <i>Pyrococcus furiosus</i>. • Specific for glyceraldehyde-3-phosphate. 	
Cross-references	
BRENDA	1.2.7.6
PUMA2	1.2.7.6
PRIAM enzyme-specific profiles	1.2.7.6
KEGG Ligand Database for Enzyme Nomenclature	1.2.7.6
IUBMB Enzyme Nomenclature	1.2.7.6
IntEnz	1.2.7.6
MEDLINE	Find literature relating to 1.2.7.6
MetaCyc	1.2.7.6

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